OCULAR MORBIDITY IN MECHANICAL INJURIES

DISSERTATION SUBMITTED TO

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M.S. DEGREE EXAMINATION OF

BRANCH III OPHTHALMOLOGY

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CERTIFICATE

This is to certify that this dissertation entitled **Ocular morbidity in mechanical injuries** submitted by **Dr. A. Subhalakshmi** to the faculty of Ophthalmology The Tamil Nadu Dr. MGR Medical University, Chennai in partial fulfillment of the requirement for the award of M.S Degree Branch III (Ophthalmology), is a bonafide research work carried out by her under our direct supervision and guidance.

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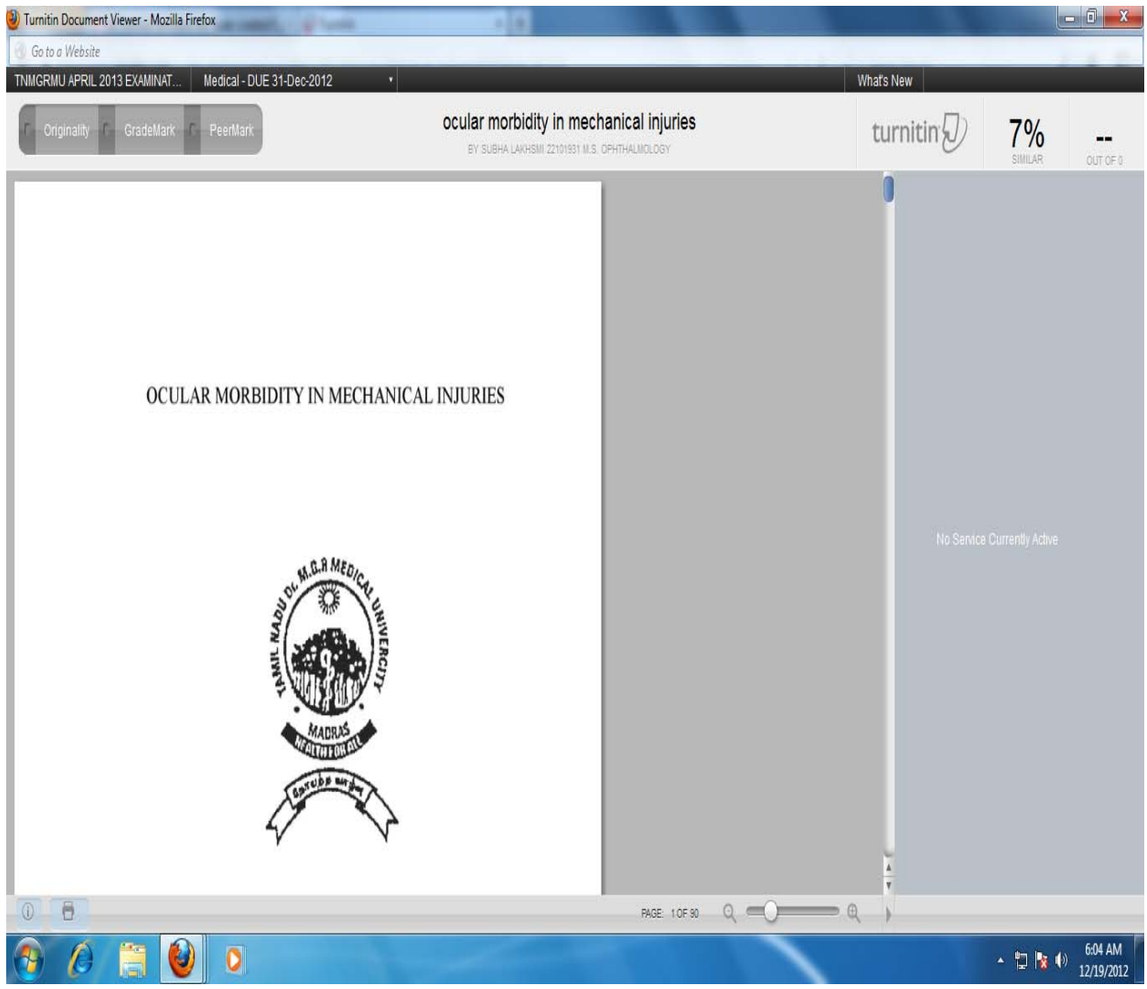
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INTRODUCTION

Mechanical injury to eye can occur in a variety of ways and produce myriad clinical sequelae. In this era of high speed traffic and industrialization, incidences of injuries are increasing in general. Like any other part of the body, eyes are also not exempt from these injuries, in spite of being protected by lids, projected margins of orbit, the nose and cushion of fat from behind.

Estimates of the incidence of eye trauma vary widely. Ocular injury due to mechanical trauma is one of the major health problems in India. Children at play in recreational activities, young men at work in urban factories, construction sites and rural agricultural settings and older people who suffer falls commonly suffer the consequences of ocular injury. The majority of injuries are sustained by active and productive individuals. Unfortunately, these injuries are often vision threatening and the lifestyle and future of the injured individual is irrevocably altered.

AIM OF THE STUDY

Aim of the study was to prospectively determine the effect of mechanical injuries on the

- Anterior segment of the eye
- Posterior segment of the eye
- Visual pathway and neuro-ophthalmic system
- Orbit and adnexa

and visual outcome of the same.

ANATOMY OF EYE

Each eyeball is a cystic structure kept distended by pressure inside it. The eyeball is not a sphere but an oblate spheroid. The anterior and posterior poles are the central points of maximal convexities of the eye ball . The equator of the eyeball lies at the midplane between the two poles.

Dimensions of adult eye ball:

Anteroposterior diameter	24 mm
Horizontal diameter	23.5 mm
Vertical diameter	23 mm
Circumference	75 mm
Volume	6.5 mm
Weight	7gm

Layers of eyeball:

Eyeball contains three layers:

Outer fibrous layer

Vascular layer (uveal tissue)

Nervous layer (retina)

Outer fibrous layer:

It's a strong outer layer, which protects the intraocular contents. Anterior $1/6^{th}$ of this fibrous layer is transparent and is called cornea. Posterior 5/6 is opaque called sclera. Junction of sclera and cornea is called limbus[1]. It is at limbus where conjunctiva is firmly attached.

Vascular layer (uveal tissue):

It supplies nutrition to eyeball. It consists of three parts:

From anterior to posterior are : iris, ciliary body and choroids

Nervous layer (retina): It's concerned with visual functions.

Segments and chambers of eyeball: The eyeball is divided into two segments:

- Anterior segment
- Posterior segment

<u>Anterior segment :</u>

It includes crystalline lens and structures anterior to it, i.e iris, cornea and two aqueous humor filled spaces- the anterior and posterior chambers.

Anterior chamber: It is bounded anteriorly by back of the cornea and posteriorly by iris and ciliary body[2]. The anterior chamber is about 2.5 mm deep in centre in normal adults and 1mm deep in periphery. It contains about 0.25 ml of aqueous humor.

Posterior chamber: It is a triangular space containing 0.06ml of aqueous

humor. It is bounded anteriorly by the posterior surface of iris and part of ciliary body, posteriorly by crystalline lens and its zonules and laterally by ciliary body**.**

Posterior segment:

It includes structures posterior to lens i.e, vitreous humor, retina, choroids and optic nerve.

ANATOMY OF ORBIT

The two bony orbits are quadrangular truncated pyramids situated between the anterior cranial fossa above and the maxillary sinuses below. Each orbit is formed by seven bones namely: frontal, ethmoid, lacrimal, palatine, maxilla, zygomatic and sphenoid.

The depth of orbit is 42mm along medial wall and 50 mm along the lateral wall. The base of the orbit is 40mm width and 35mm height.

Orbital index= height/width x 100	Orbital index shows racial variation
Orbital index >89	Megasenes (eg. Orientals)
Orbital index between 83-89	Mesosenes (eg. Caucasians)
Orbital index <83	Microsenes
Volume of orbit	29ml

WALLS OF ORBIT

Medial wall:

The medial wall of orbit is quadrilateral in shape and is formed by the frontal process of the maxilla, the lacrimal bone, the orbital plate of ethmoid bone and the body of sphenoid bone.

Relations:

Medial to medial wall lie anterior ethmoidal air sinuses, middle meatus of nose, middle and posterior ethmoidal sinuses and sphenoidal air sinus.

The orbital surface of the medial wall is related to superior oblique muscle in the upper part near the roof and medial rectus muscle in the middle part. Anterior ethmoidal nerve, posterior ethmoidal nerve, infratrochlear nerve and terminal branch of ophthalmic artery lie between these two muscles.

Inferior wall(floor of orbit):

The floor of orbit is triangular in shape. The orbital surface of the maxillary bone medially, the orbital surface of the zygomatic bone laterally and the palatine bone posteriorly form the floor. Inferior orbital fissure separates the posterior part of floor from lateral wall.

Relations:

Below = maxillary air sinus and palatine air

ells.

Above = inferior rectus muscle, inferior oblique muscle and nerve to inferior oblique.

Lateral wall:

It is triangular in shape, formed anteriorly by zygomatic bone and posteriorly by greater wing of sphenoid bone.

Relations:

Laterally—It separates the orbit from temporal fossa anteriorly and from middle cranial fossa posteriorly

Medially—related to lateral rectus, lacrimal nerve and vessels, zygomatic nerve and the communication between zygomatic and lacrimal nerves.

Roof:

It is triangular in shape and is formed mainly by the orbital plate of frontal bone. Behind this it is formed by lesser wing of sphenoid bone.

Relations:

Above: Frontal lobe of cerebrum and meninges

Below: periorbita, frontal nerve, levator palphebrae superioris, superior rectus, superior oblique, trochlear nerve and lacrimal gland.

At the junction of the roof and the medial wall are anterior and posterior ethmoid canals.

At the junction of roof and lateral wall is a gap posteriorly, the superior orbital fissure

Base of orbit:

The anterior open end of the orbit is referred to as base. It is bounded by orbital margins.

Superior orbital margin- formed by orbital arch of frontal bone

Lateral orbital margin- by zygomatic process of frontal bone and zygomatic bone.

Inferior orbital margin- by the zygomatic bone laterally and maxilla medially

Medial orbital margin- below by anterior lacrimal crest on the frontal process of maxilla and above by frontal bone.

Apex of orbit:

It is the posterior end of the orbit. Here the four orbital walls converge. The apex has two orifices- the optic canal and the superior orbital fissure[3]

MECHANICAL INJURIES TO EYE

The Ocular Trauma Classification Group has attempted to develop a uniform classification system for mechanical injuries to the eye, based on primary evaluation.

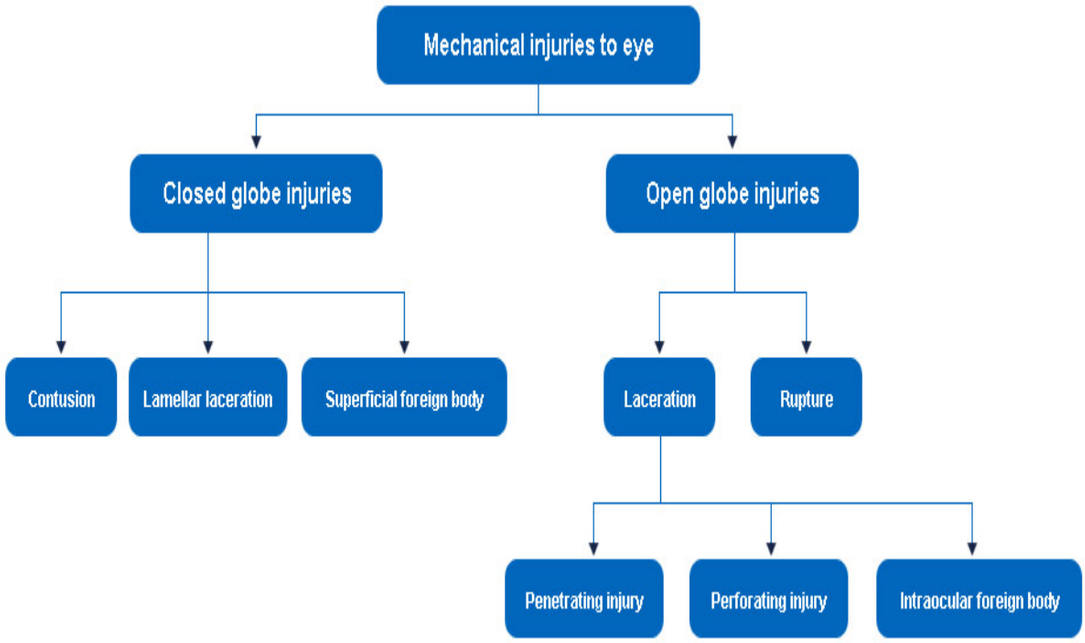
Injuries are broadly divided into two categories

A. open globe – full- thickness defects in the corneoscleral coat of the

eye

B. closed globe – ocular injury without a full – thickness defect of the

coats.



Open globe injuries:

It is further subdivided into

1. ruptures of globe with an inside to outside break in the ocular coats.

2. laceration is used to denote full thickness outside to inside break in ocular coats.

This can be further sub – divided into

a. penetrating injury -if the object traverses the coat only once

b. perforating injury -if both an entry and exit wound are present.

Modes of open globe injuries:

a. Trauma by sharp and pointed instruments like needles, knives, nails, arrows, screw-drivers, pens, pencils, compasses, glass pieces and so on.

b. Trauma by foreign bodies traveling at high speed such as bullet injuries and iron foreign bodies in lathe workers.

Closed globe injuries:

It usually follows blunt trauma. It is subdivided into:

1. contusion or concussion injuries
2. Superficial foreign bodies are frequently found on the cornea and conjunctiva
3. Lamellar laceration – partial thickness injury of the coats.

- Modes of closed globe injury:

a. Direct blow to the eye ball by fist, ball or blunt instruments like sticks and big stones

b. Accidental blunt trauma to eyeball may also occur inroad side accidents, automobile accidents, injuries by agricultural and industrial instruments / machines and fall upon the projecting blunt objects[4]

- Mechanics of closed globe injury: Blunt trauma of eye ball produces damage by different forces.

  a. Direct impact on the globe- It produces maximum damage at the point where the blow is received

  b. Compression wave force- It's transmitted through the fluid contents of eyeball in all directions and strikes the angle of anterior chamber, pushes the iris lens diaphragm posteriorly and also strikes the retina and choroid. This may cause considerable damage. Sometimes the compression wave may be so explosive that the maximum damage may be produced at a point distant from site of impact. This is called contre- coup damage.

  c. Reflected compression wave force: After striking the outer coats the compression waves are reflected towards the posterior pole and may cause foveal damage.

  d. Rebound compression wave force: After striking the posterior wall of the globe, the compression waves rebound back an anteriorly, which damages the retina and choroids by forward pull and lens- iris forward thrust from back[5]

  e. Indirect force: Ocular damage may also be caused by the indirect forces from bony walls and elastic contents of the orbit, when globe strikes against these structures.

For descriptive purposes, mechanical injuries can be classified broadly into:

1. Anterior segment injuries
2. Posterior segment injuries
3. Neuro ophthalmic manifestations of trauma
4. Orbital and adnexal injuries

ANTERIOR SEGMENT INJURIES

Anterior segment trauma is a common cause of ocular morbidity and constitutes an important segment of ophthalmic practice.

A. Subconjunctival haemorrhage:

Blood under conjunctiva has a fiery red appearance. Inability to discern the posterior limit of subconjunctival haemorrhage indicates orbital or intracranial injury. It is managed by reassurance and further imaging to rule out serious injury.

B. Superficial foreign bodies:

Foreign body may be present in the superior tarsal sulcus. It is detected by eversion of upper lid. Double eversion using Desmarre retracter is necessary to reveal foreign bodies in deep fornices.

Iron foreign body may embed in corneal stroma, when a rust ring may be formed. A corneal foreign body may be removed under topical anaesthesia at the slit lamp using foreign body spud or 25 guage needle. After foreign body removal, topical anaesthetic drops are applied hourly, and pressure patch applied if epithelial abrasion is significant. Patient are closely monitored till epithelium heals.

C. Corneal abrasion:

It is a break in corneal epithelium due to minor trauma which stains with fluorescein. Hourly broad spectrum topical antibiotics, cycloplegics and pressure patch ensure healing within 24 hours.

Damage to epithelial basement membrane adhesion complexes may lead to recurrent corneal erosions. This causes acute onset of pain on awakening . Signs vary from irregular area of raised epithelium causing focal break in tear film to a full thickness epithelial defect with elevated grey margins.

It is treated by epithelial debridement, pressure patch, copious lubricants for 6-8 weeks, bandage contact lens for 2 months or intrastromal punctures or photo therapeutic keratectomy.[6]

D. Hyphema:

It is common in young males , usually due to injury to peripheral iris and anterior ciliary body in the form of iridodialysis, cyclodialysis, disruption of major arterial circle, arteries of ciliary body and choroidal vessels.

Raised IOP, vascular spasm and formation of fibrin clot can stop the hyphema. The clot may have a bilobed configuration with contiguous blood in anterior and posterior chamber called 8 ball hyphema.

Fibrin degradation products are then cleared through the normal trabecular outflow pathways and by iris vessels to a small extent

Grades of hyphema-

Grade I - blood level less than 1/3 of anterior chamber (incidence >50%) Grade II - blood level 1/3 to ½ of anterior chamber

Grade III - blood level ½ to near total

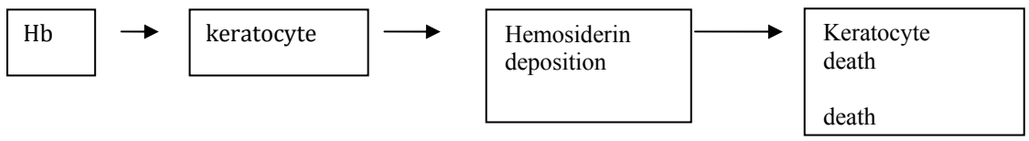
Grade IV blood occupying total anterior chamber (incidence < 10%)

A complete eye evaluation is required to assess concomitant injury and occult globe rupture. Dilated fundoscopy is done at the time of presentation. Vision, hyphema status and IOP are documented in every follow up visits. Scleral depression is avoided upto one month to avoid rebleeding.

Rebleeding occurs commonly between 2 to 5 days after trauma. It is indicated by the presence of bright red layered blood over clotted blood and increased size of hypema. It indicates poor pronosis.

Corneal blood staining and optic neuropathy due to persistent hyphema and elevated IOP lead to permanent visual loss .

(i) Corneal blood staining occurs due to combination of elevated IOP, endothelial dysfunction and anterior chamber blood.



Slit lamp examination shows yellow granular changes and stroma shows reduced fibrillar definition.

(ii)Optic neuropathy- occurs if IOP is persistently elevated > 35 mm of Hg for 7 days. So to prevent optic neuropathy, intervention should be done if IOP > 35 mm of Hg.

Hyphema is best managed:

Medically by

a) 1% Atropine eye drop 3 times a day- gives comfort in the setting of iritis

b) 1% prednisolone acetate eye drop – helps to minimize discomfort in iritis

c) Antifibrinolytic agents – Aminocaproic acid 50mg/ kg orally every 4hrs

upto 30g/day for 5 days. It competitively inhibits conversion of plasminogen to plasmin, which degrades fibrin clot.

- Surgical management- 5% of patients may require anterior chamber wash out, usually performed through two clear corneal incision. As the clot retracts in 4-7 days, the ideal time for AC wash is 7 days.

Other criteria for early surgical intervention are:

1. IOP > 50mm of Hg for 5 days

2. IOP > 35mm of Hg for 7 days to prevent optic nerve damage

3. IOP > 25mm of Hg for 5 days in total or near total hyphema to prevent

corneal blood staining

4.IOP averages >25mm of Hg for 24 hours in patients with sickle haemoglobinopathies

Sickle cell anaemia patients with hyphema should be managed aggressively. Sickle cells are elongated and rigid that reduces their egress through trabecular outflow pathways. Even small hyphema can clog the trabecular meshwork leading to raised IOP. Consequent hypo perfusion can lead to anterior segment hypoxia, acidosis and hypercarbia, thus encouraging sickling. These patients are also predisposed to optic nerve damage and central retinal artery occlusion with a marginal raise in IOP. Therefore, IOP should be monitored and treated aggressively by topical beta adrenergic antagonists and AC wash out. Carbonic anhydrase inhibitors are avoided.

Approximately, 75% of patients with hyphema regain visual acuity of greater than 20/50. Long term visual loss in hyphema is more commonly due to traumatic cataract, posterior pole inury and optic nerve damage than to complications related directly to blood itself.

E. Iris injury

- Tears of iris sphincter and pupillary frill are frequent and may cause an irregular unreactive and chronically dilated pupil.
- Dialysis of the iris root produces a D shaped pupil and may result in acute hyphema.
- Damage to trabecular meshwork and formation of peripheral anterior synechiae may lead to elevated IOP[7].
- Less frequently, a cyclodialysis may occur leading to chronic hypotony.

Management:

- Sunglasses may decrease glare and photophobia. Tinted contact lenses or those with an artificial pupil may decrease these symptoms
- The argon and Nd YAG lasers can be used to recenter an eccentric pupil. Laser sphinterotomy may open an eccentric or secluded pupil toward visual axis.

- Mc Cannel suture using 10 0 polypropylene to suture iris dialysis[8]

F. Traumatic glaucoma:

(i)Immediate glaucoma may be caused by clogging the trabecular meshwork by red blood cells or inflammatory cells. Patients with large hyphema may have pupillary block and acute angle closure [9].

(ii) An intermediate term glaucoma may be caused by ghost cell glaucoma occurring 3-4 weeks after vitreous haemorrhage. Rigid khaki coloured non pliable ghost red blood cells may percolate forward and clog the trabecular meshwork.

(iii)Late glaucoma –

a. frequently associated with angle recession. It is identified by, broadened ciliary band by gonioscopy, AC appearing abnormally deep and fibrotic changes in the angle

b. pupillary block

c. peripheral anterior synechiae

d. scarring of trabecular meshwork

e. injuries to lens – phacomorphic, phacolytic and phacoanaphylactic

glaucoma.

Management

1. Topical corticosteroids are useful to minimize inflammation, prevent

formation of anterior and posterior synechiae and prevent scarring of trabecular meshwork

2. Acute IOP elevations treated medically with aqueous suppressants

3. In cases of pupillary block, laser peripheral iridotomy, hyphema wash out or lens surgery may be necessary to treat acute IOP elevations

4. Chronic glaucoma should be treated with medical therapy.

5. In angle recession glaucoma- laser trabeculopasty has low success rate due to intrinsic damage to trabecular meshwork.

6. Peripheral anterior synechiae- Laser gonioplasty may be useful. Filtration surgery needed if the above said measures fail to control IOP.

G. Lens injury:

Blunt trauma to eye may lead to contusion cataract and lens subluxation or dislocation. Rupture of capsule lead to rapid opacification of lens. Distortion of the globe with equatorial scleral expansion may also cause capsular rupture or result in zonular dehiscence with consequent lens subluxation or dislocation.

a. Contusion cataracts – patient presents with deterioration of visual acuity.

b. Complete lens dislocation into vitreous render the eye functionally aphakic

c. Zonular dehiscence may be signalled by iridodonesis and phacodonesis. In addition anterior chamber may show prolapsed vitreous. Lens capsule should be examined for anterior or posterior capsule rupture. Special attention should be devoted for lens induced IOP elevations and inflammation.

d. Pupillary block may occur with subluxated or dislocated lens or with an intumescent cataract following capsular rupture. Release of lens proteins may cause subsequent phacolytic glaucoma secondary to high molecular weight proteins, lens particles and macrophages clogging the aqueous outflow channels. Such findings usually suggest immediate surgical intervention.

Management:

The anatomic position and stability of the lens dictate the method by which it is managed surgically.

(i) If the capsule and zonules are intact or minimally subluxated a standard extracapsular extraction or phacoemulsification procedure can be performed.

(ii) If zonular damage is significant but lens is free of vitreous, intracapsular extraction may be performed through the limbus[10]

(iii) If vitreous has prolapsed around the lens, vitreous must be first removed. Extreme caution must be taken not to exert vitreous traction during lens delivery.

(iv) In cases with significant vitreous disruption and posterior lens dislocation, parsplana vitrectomy- lensectomy approach is preferred.

The placement of an IOL, selection of IOL type and implantation technique must be tailored to the individual case. If posterior capsule is intact without vitreous loss or undue anterior segment disruption, then posterior chamber IOL can be implanted. In the absence of posterior capsule support flexible anterior chamber IOL can be implanted. Transcleral sutured posterior chamber lenses or glued IOLs can be used if adequate vitrectomy is performed.

H. Corneoscleral lacerations

a. Cultures should be taken from

1.wound margins

2. a portion of any excised tissue

3.intraocular foreign bodies

Specimens are inoculated directly into blood and chocolate agar plates, fluid thioglycolate broth or cooked meat broth from anaerobes, as well as on Sabouraud's medium for fungus.

b. Intravenous antibiotic started immediately. Broadspectrum coverage is afforded by a combination of a cephalosporin or vancomycin for gram positive coverage and an aminoglycoside for gram negative coverage. Topical antibiotics are better avoided in open globe injury. If the wound is self sealing with minimal uveal prolapse, a topical antibiotic is administered.

c. Surgical management- The primary objective of initial surgery is complete water tight closure of the globe. Secondary goals include removal of disrupted lens and vitreous, avoidance of uveal and vitreous incarceration in the wound, removal of intraocular foreign bodies and restoration of normal anatomical relationship.

d. Non perforating corneal lacerations All suspected non perforating corneal lacerations should be carefully examined to rule

out occult perforation of Descemet`s membrane. Siedel `s test is performed with 2% fluorescein to check for microscopic leaks.

(i) Small perforating injuries may self seal. When the wound is somewhat unstable , bandage contact lens can be used to support the wound and promote reepithelialisation by shielding the wound from lid movement. For small self sealing corneal lacerations, a bandage contact lens may be sufficient to protect and support the wound as it heals, upto 3 to 6 weeks. Topical antibiotics and cycloplegics are used with bandage contact lens in place.

(ii)Long partial thickness wound and in those with significant wound override or gape need suturing.

(iiv)Puncture wounds with small amount of central tissue loss and for wounds that require suture placement in visual axis, cyanoacrylate tissue adhesive is applied under topical anaesthesia at a slit lamp or under operating microscope. Immediately after the adhesive has adequately dried bandage soft contact lens is applied.

(iv) Large lacerations (greater than 2 to 3 mm) , displaced wounds, wounds with loss of corneal tissue and laceration with accompanying iris or lens incaraeration must be brought to the operating room for definitive surgery and suture placement. Surgery is done under general anaesthesia if there is no medical contraindication. If the wound is relatively water tight and the anterior chamber formed, it

may be sutured directly. The ultimate aim of corneal suturing is definitive placement of corneal sutures to make the wound watertight, minimize scarring and reconstruct the native nonastigmatic corneal contour. Monofilament 10-0 nylon sutures are used, sutures approximately 90% deep in stroma and of equal depth on both sides of the wound. The wound should be checked for leaks by gentle pressure on the globe with dry cellulose sponge.

(v) Corneal laceration with iris incarceration After an extensive corneal laceration anterior chamber shallows , with consequent iris incarceration or prolapse. If prolapsed the tissue must be critically evaluated . . Healthy appearing tissue may be reposited.

Obviously devitalized tissue should be exised. If foreign body is present within the tissue it is certainly excised. Moreover the tissue should be evaluated for surface epithelialisation. If it is present, it may lead to seeding of anterior chamber leading to epithelial proliferation. Iris reposition done by direct sweeping with a spatula or irrigating canula through the paracentesis site, followed by suturing of the corneal laceration**.**

(vi) Corneal laceration with Lens involvement: Lens disruption is difficult to diagnose due to fibrinous anterior chamber reaction, pupillary membrane and corneal edema. It is better to intervene after treating corneal edema.

A lens with a disrupted capsule and flocculent capsular material liberated into the anterior chamber is preferably removed to prevent ongoing phacogenic postoperative inflammation. If the lens is clearly cataractous it may be removed in the initial operation. However a stable lens with intact capsule and without liberated cortical material can be treated in secondary procedure.

(vii)Corneal laceration with vitreous involvement: Violation of vitreous face usually accompanies corneal laceration with lens involvement. The primary goal is to relieve any vitreous incarceration in the traumatic or surgical wounds. It is important to remove disrupted vitreous from anterior chamber to minimize subsequent corneal endothelial damage. Preferably automated microvitrectomy instrumentation is used. After vitrectomy, dry cellulose sponges are useful to identify vitreous strands remaining in the anterior chamber or at laceration site. At the conclusion of surgery, the pupil should be round without peaking, wound free of incarceration and the vitreous behind the iris.

(viii)Simple corneoscleral laceration: Lacerations extending beyond the limbus into sclera need to be explored to delineate the full extent of the wound. For large laceration with unstable globe, sutures may be placed before definitive exploration of the wound to restore structural integrity. The limbus is first approximated to restore correct anatomic

relationship. The prolapsed iris is next reposited and corneal wound is closed. The extent of scleral laceration is then ascertained. The laceration occasionally extends far posteriorly, sometimes upto optic nerve. In these cases with poor prognosis, it is probably preferable to leave the more posterior extent of the wound unsutured than to distort the globe excessively.

(ix)Corneoscleral lacerations with uveal and vitreousprolapse: Vitreous prolapsing through a scleral wound is easily secured with dry cellulose sponges and cut flush with the sclera. Care is taken to avoid traction on vitreous. A vitrectomy instrument can be used to excise vitreous from wound.

Uveal tissue often prolapses from gaping scleral lacerations. In severe cases prolapsed tissue is excised to allow for wound closure. Any tissue removed should be identified histolgically. Evidence of retina in excised portion indicates poor prognosis.

The preferred method for scleral closure over prolapsed uvea is a zippering technique. The wound is closed from the anterior limbal end with interrupted sutures, that are successively placed posteriorly. The sutures are relatively closely spaced, with the goal to oversew the uveal tissue with the sclera.

I. Anterior segment intra ocular foreign bodies: Although search for an intraocular foreign body must be made in all eyes with penetrating

injuries, foreign bodies are more common after activities involving striking metal on metal.

An entry wound through cornea may be small and self sealing, and the one through sclera may be obscured by subconjunctival haemorrhage. Anterior segment intraocular foreign body may lodge in the angle and is signaled by, focal corneal edema overlying an angle in that area. Gonioscopy can be performed to confirm and localize the foreign body. A foreign body may embed on the lens and be seen as focal cataract formation. Radiographic examination, including plain radiographs, CT scanning and MRI may be helpful in detecting an intraocular foreign body.

Foreign body in the angle or in the iris or in the lens can be removed through a limbal incision placed either directly over the object or across the anterior chamber. In some cases of metallic foreign body, magnet can be used for extraction

Anterior segment reconstruction:

Despite meticulous primary repair of a penetrating corneoscleral injury, normal wound healing mechanisms may lead to devastating late complications in eye trauma. Such sequelae include

- corneal scarring, astigmatism
- glaucoma

- Vitreous incarceration within ocular wounds and associated chronic inflammation – cystoid macular edema, retinal detachment, infection.
- Pupillary or cyclitic membrane
- conjunctival scarring and symblepheran
- ocular surface epithelial damage leading to persistent epithelial defects, stromal vascularisation, sterile stromal ulceration

Procedures done are- Lensectomy is frequently combined with open sky vitrectomy.

- Goniosynechiolysis
- Reconstructive iridoplasty
- Membrane removal and iridoplasty should be done before donor cornea is placed and sutured.

2. POSTERIOR SEGMENT TRAUMA

It includes any changes induced in the eye by injury that affects the vitreous body, retina, choroids, optic nerve and sclera. The injury could be due to direct effect of blunt trauma, perforating trauma, electromagnetic trauma or posterior segment sequelae of remote trauma.

A. Scleral rupture:

The sclera is resistant to severe injury but with severe blunt injury rupture can occur.

a. Direct rupture is uncommon.

b. Indirect rupture occur at the site away from the site of impact in an area of scleral weakness.[11]

Most scleral ruptures are solitary but multiple ruptures can occur. The most common sites of rupture are the superior nasal and superior temporal quadrants between coreneal limbus and equator.

Scleral ruptures are occult and not easily documented by slit lamp examination. Signs of occult scleral rupture are visual acuity of light perception or no light perception, reduced ductions, ocular hypotony, hyphema, severe chemosis and bullous subconjunctival haemorrhage. Ophthalmoscopy may be limited by media opacities including hyphema, cataract and vitreous haemorrhage. Ultrasonography and computed tomography may show a shrunken globe but actual rupture is not reliably demonstrated.

The only definitive way to rule out scleral rupture is by careful, controlled exploration of globe. Eyes with scleral rupture is often severely injured internally, with choroidal and retinal tearing, suprachoroidal haemorrhage, vitreous haemorrhage, teraing of ciliary body and avulsion of optic nerve. Internal ocular injuries associated with scleral rupture make ocular reconstruction and visual

rehabilitation challenging. Success is usually measured by globe salvage and ambulatory vision . Early primary repair is an important first step in management. Vitrectomy is effective in dealing with intraocular haemorrhage with scleral rupture.

Scleral perforations include single perforations, double perforations multiple perforations accompanied by retained intraocular foreign body. It usually involves severe trauma to the lens, choroids, ciliary body and retina. Profound visual loss is often the unfortunate result of scleral perforation, despite wound closure, vitrectomy , scleral buckling and use of vitreous substitutes.

B. Choroid rupture:

The choroid is susceptible to rupture from the effects of blunt trauma applied to the globe. The rupture may occur

a. directly, at the site of application of the force

b. indirectly on the opposite side of the globe.

The choroids is susceptible to rupture because of inelastic characters of Bruch`s membrane.

Posterior choroidal ruptures are curvilinear, concentric to optic nerve and may be accompanied by intraretinal and subretinal haemorrhages. Some posterior choroidal ruptures are subtle and can be diagnosed only with the aid of fluorescein angiography. Early hypoflurescence

from choriocapillary injury and late hyperfluorescence in a curvilinear pattern concentric with optic disc are characteristic.

Choroidal ruptures at the site of direct application of force often occur in infero temporal quadrant, because the bony orbit doesn't protect the eye well in that quadrant. When choroidal ruptures are accompanied by retinal rupture, it is called retinitis sclopetaria, chorioretinitis sclopetaria or chorioretinitis plastica sclopetaria. Severe subretinal fibrosis with complete atrophy and disruption of choroid, retinal pigment epithelium and retina occur.

C. Traumatic retinal breaks

This occur when the vitreous is violently shifted away from retina. Any area of strong vitreoretinal adhesion is likely to be the site of retinal tearing. The vitreous base, lattice degeneration, previous chorioretinal scars, the fovea and retinal blood vessels are common sites of retinal tear.

Direct or coup forces applied to the retina can result in retinal necrosis and atrophy. Often massive chorioretinal scarring occur at the margins of the retinal atrophy after the haemorrhage clears. As a result , retinal detachment does not occur.

All nonfoveal traumatic retinal tears deserve treatment with cryotheraphy, laser photocoagulation or scleral buckling unless chorioretinal scarring adequately surrounds the tear.

D.Retinal dialysis

It is the most common traumatic retinal break, refers to a retinal break occurring at the anterior edge of ora serrata and posterior edge of which is attached to the vitreous base. Inferior temporal and superior nasal quadrants are the most common areas for retinal dialysis.

The retina and vitreous are tightly adherent at the vitreous base and as the vitreous base is avulsed into vitreous cavity retina follows, creating a tearing of retina at or near ora serrata.

The challenge of management of retinal dialysis is early recognition. Accurate diagnosis requires skill at indirect ophthalmoscopy with scleral depression. If retinal dialysis without retinal detachment is observed, cryotherapy or laser prophylactic therapy is indicated. If retinal detachment is present, scleral buckling achieves a reattachment rate of 98%

E.Traumatic retinal detachment:

Its commonly seen more frequently in young male patients. Traumatic retinal detachment is associated with superior nasal dialysis in 22%, inferior temporal dialysis in 31%, giant retinal tears in 16%, retinal flap tears in 11% and tears associated with lattice degeneration in8%

Traumatic retinal detachments are managed by pneumatic retinopexy , scleral buckling or vitrectomy depending on the retinal breaks and degree of proliferative vitreoretinopathy.

F. Commotio retinae:

Although the retina may be torn during blunt trauma, severe nontearing injury can also occur in a coup or contre coup location. The term applied to blunt nontearing retinal injury is commotio retina or Berlins edema. Commotio retina has a whitish gray appearance with a cherry red spot that can be observed several hours after blunt trauma and is occasionally accompanied by intra retinal haemorrhage retinal pigment epithelial detachment or choroidal rupture. Visual acuity is very much reduced or may be unaffected. No effective treatment for commotio retina is known.

G.Vitreous changes in blunt trauma:

The vitreous can be injured in blunt trauma by disinsertion or opacification.

a. Disinsertion occur at vireous base, optic nerve, retinal vessels, fovea, lattice degeneration and chorio retinal scars. The retina is usually severely injured and dominates the clinical picture. The avulsed vitreous base has the appearance of a loose clothline, a

hammock or a ribbon suspended loosely through vitreous cavity. It is pathognomonic of blunt trauma.

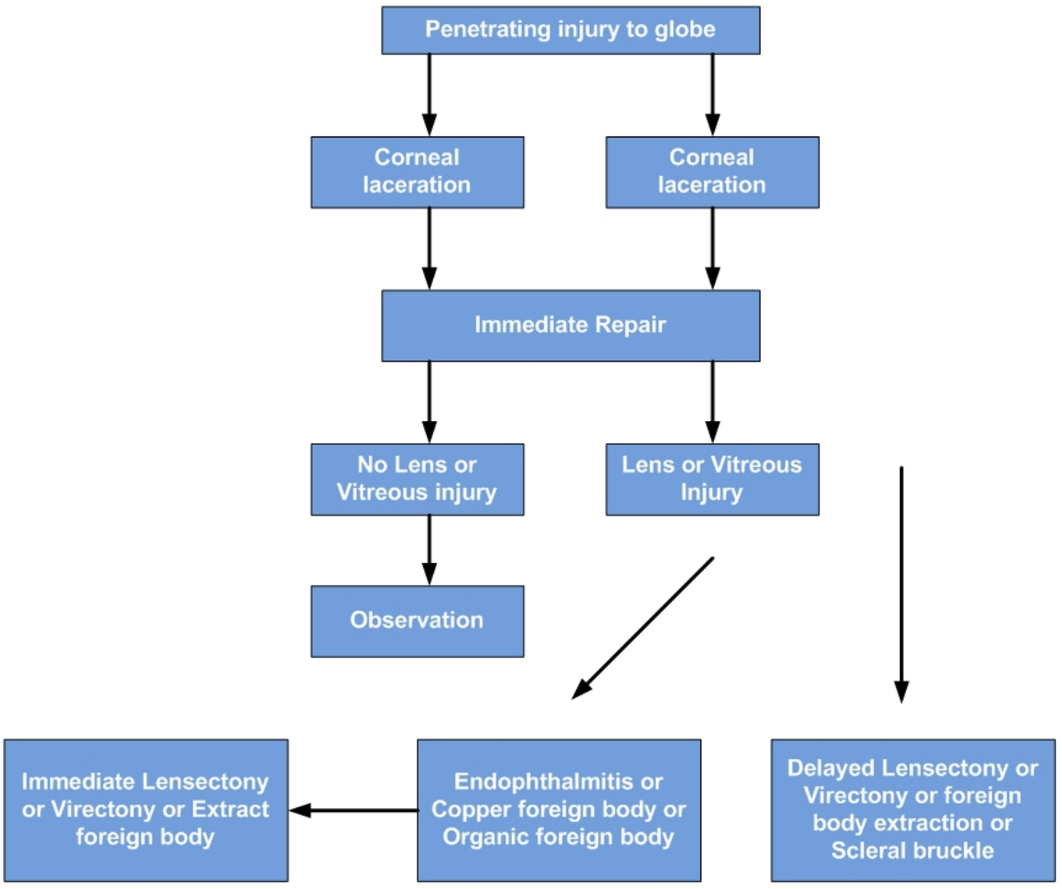
b. Vitreous opacification usually results after haemorrhage from torn retinal, choroidal or ciliary body vessels. Most vitreous haemorrhage clear spontaneously but eyes with dense haemorrhage may need vitrectomy to clear the visual axis. Observation for upto six months may be rewarded for spontaneous clearing and avoidance of intraocular surgery. The goal of surgery in a severely lacerated globe is prevention of secondary complications such as endophthalmitis, retinal detachment, cyclitic membrane and toxic injury from a retained foreign body.

Repair of posterior segment laceration is divided into primary and secondary repair.

a. Primary repair refers to immediate efforts to restore the external anatomic integrity of the globe. It should be attempted as soon as possible after injury. Ocular surgery may be delayed by associated life threatening injuries to the head, chest or abdomen.

b. Secondary repair refers to surgical steps taken to restore anatomical integrity of the eye. Cataract extraction, drainage of haemorrhagic choroidal detachment, vitrectomy, removal of an intraocular foreign body, injection of intraocular antibiotics and scleral buckling may be performed.

The goal of vitrectomy is complete removal of the injured vitreous gel, including the posterior vitreous surface.



H. Traumatic endophthalmitis:

Posttraumatic infectious endophthalmitis occurs in 2 to 7% of all penetrating intraocular injuries and in 7 to 13 % of retained intraocular foreign bodies. Eyes at high risk are those injured by

foreign objects contaminated by soil or vegetable matter. Staphylococcus and Bacillus species are the most causes of posttraumatic endophthalmitis.

I. Intraocular foreign body

Penetration of the globe with foreign bodies are frequent. Seriousness of such injuries depend on the retention and toxicity of the intraocular foreign body[12]

Foreign bodies types: 1. Metallic –a. magnetic (eg) iron

b. non magnetic (eg) copper

2. Non metallic – plastic, wood

Ion poisoning of intraocular epithelial structures is seen with copper, iron, lead zinc and nickel. In general, metals with low redox potential such as iron and copper have the greatest potential for metallosis.

Modes of damage:

a. mechanical effects – depends on size, velocity and type of foreign body. Foreign bodies of size greater than 2mm cause extensive damage

b. introduction of infection- pieces of the wood and stones carry a greater chance for infection

c. reactions of foreign body- depends on chemical nature of foreign body. It can be of four types.

(i) no reaction- usually with inert substances like glass, plastic

(ii) local irritative action leading to encapsulation of foreign body

(iii) suppurative reaction is excited by copper, mercury particles, nickel , zinc etc

(iv)specific reaction – iron causing siderosis bulbi, copper alloys producing chalcosis

Management of intraocular foreign body:

To come to a correct diagnosis following steps are followed:

1. History
2. Thorough ocular examination
3. X ray orbit antero-posterior and lateral views
4. Localisation of foreign body by CT scan, ultrasound

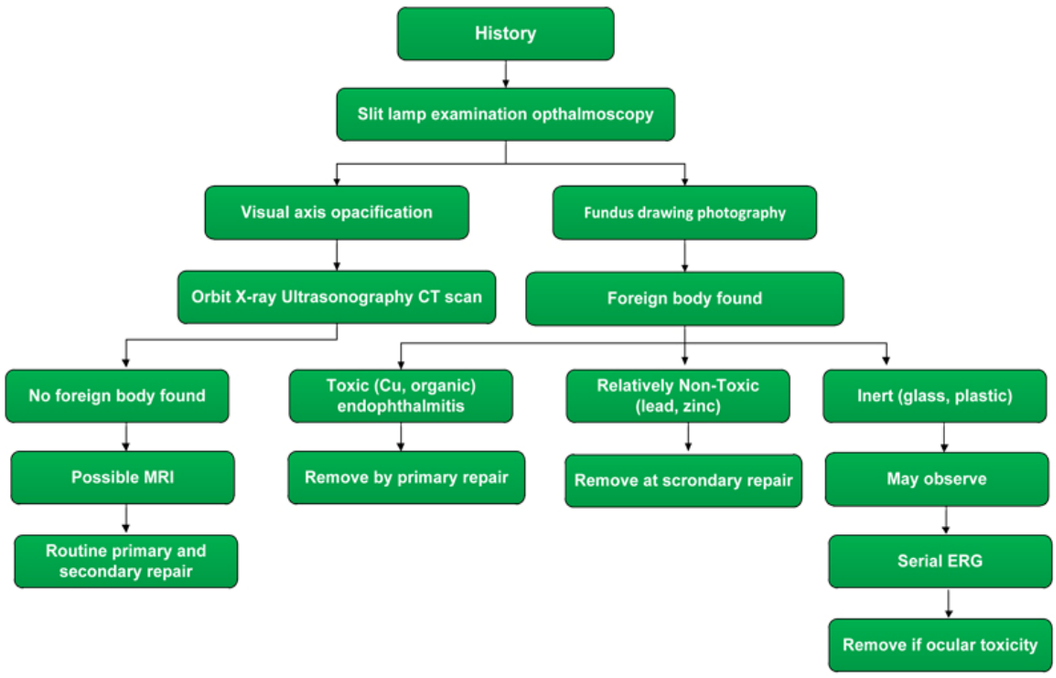
Treatment: IOFB should always be removed except when it is inert and probably sterile or when little damage has been done to the vision and the process of removal may destroy sight. Method of removal depends on the site of lodgement of the foreign body

1. FB in anterior chamber: removed through a corneal inciscion directed towards
2. FB entangled in iris tissue – removed by performing sectoral iridectomy of the part containing the FB
3. FB in the lens: ECCE with intraocular lens implantation done
4. FB in the vitreous and retina – removed by posterior route

Magnetic FB- by pars plana sclerotomy using forceps or external magnet

Non magnetic FB- removed by forceps with parsplana vitrectomy

Algorithm of management of intraocular foreign body



3. NEUROPHTHALMIC MANIFESTATIONS OF TRAUMA

A.Optic nerve injury:

The clinical manifestations of optic nerve trauma include

(i) decreased visual acuity

(ii) visual field defects

(iii) dyschromotopsia

(iv) relative afferent pupillary defect

(v) occasionally fundoscopic abnormalities of optic disc.

In patients with visual complaints, the visual acuity range from normal to perception of light. Patients with normal acuity may have visual field defects that spare fixation. Damage to optic chiasma may occur secondary to blunt trauma. Therefore careful examination of visual field in the fellow eye will detect such lesions.

Trauma to optic nerve may be direct or indirect.

a. Direct injury commonly occurs in the form of penetration injuries or severe blunt trauma to the orbit and globe. Laceration or transection of the nerve may occur when a variety of objects and projectiles penetrate the eye, orbit or cranium. In these cases, loss of vision is immediate and usually complete. Tractional forces on the globe may cause avulsion of optic nerve head. Retained orbital foreign bodies or fragments of orbital fractures may impinge on optic nerve.

Optic nerve sheath haemorrhage and traumatic arachnoid cysts may also compress the optic nerve. Increased tissue pressure caused by blood or air within the confines of orbit may compromise visual functions by optic nerve compression and possibly vascular perfusion

b. Indirect traumatic optic neuropathy is defined as traumatic loss of vision that occurs without external or initial ophthalmoscopic evidence of injury to the optic nerve. Loss of vision may be

immediate or delayed. The optic disc may appear normal or edematous, depending on whether the injury involves the posterior or anterior portions of nerve respectively[13]. Optic atrophy can be identified within one week in anterior lesions and in 3-6 wk in posterior lesions.

Management:

a. High dose intravenous methylprednisolone is effective in reducing the morbidity due to optic nerve trauma. The rationale for corticosteroid treatment is reduction of tissue edema, resulting in increased vascular perfusion. Additionally, membrane stabilization is thought to reduce tissue necrosis.

A reasonable regimen would be IV methyl prednisolone in a 1g loading dose, followed by 250- 500 mg every 6hrs for 3-5 days. Medical treatment must be instituted as soon as the diagnosis of traumatic optic neuropathy is confirmed clinically. Following institution of treatment, the patient is observed for approximately 48-72 hrs. If visual function improves on corticosteroid therapy, conversion to a tapering course of oral therapy after 48 hrs. If there is no response, optic canal decompression is considered. Required

b. Surgical intervention is needed when there is delayed loss of vision while the patient is receiving corticosteroids or during tapering of corticosteroids which implies a compressive lesion. It involves

decompression of optic nerve at the level of optic canal. Optic nerve sheath fenestration near the neuroocular junction is recommended for an intrasheath optic nerve hematoma. Timely decompression of such hematomas by lateral canthotomy or direct drainage may be a sight saving maneuver.

Surgical decompression of optic canal is indicated if neuroimaging confirms the presence of bone fragments or foreign bodies impinging on optic nerve.

B. Chiasmal lesions

Traumatic lesions of optic chiasm occur but are uncommonly treated, because the trauma that produces such lesions are usually fatal. Mechanisms for causing damage to the optic chiasm include:

a. disruption of the chiasmal blood supply by shearing, thrombosis or spasm of nutrient vessels.

b. Penetrating injuries or fractures may result in laceration or tearing of the chiasm.

c. Compression of the chiasm may occur by herniation of the gyrus rectus or by a dilated third ventricle secondary to hydrocephalus. Associated damage to the hypothalamic-pituitary axis, resulting in diabetes insipidus and multiple cranial nerve palsies may be present.

There is no effective treatment for optic chiasmal trauma. Primary optic atrophy is usually noted 6-8 wks following injury.

C. Retrochiasmal lesions:

Lesions of the posterior visual pathway are not uncommon and may involve the optic tract, geniculocalcrine pathway, occipital cortex or surrounding visual association areas. Patients may complain of blurred vision in a portion of their visual field.

All retrochiasmal lesions produce homonymous visual field defects. Generally more anterior the lesion, more incongruous will be the field defects. Lesions placed more posteriorly produce congruous field defects. Cortical blindness is a condition that occurs when there is bilateral involvement of occipital visual cortex. Most common cause of cortical blindness is a penetrating injury of the occiput like gunshot wound that result in destruction of brain tissue. Less commonly, closed head injury may cause cortical blindness. This usually results from blow of concussive force to occiput. Visual loss may be immediate or delayed. These patients may experience a gradual return of vision without permanent sequelae.

Neuroimaging can be extremely valuable in demonstrating the extent of these lesions, but clinical examination gives more clues.

D. Oculomotor nerve injury

Traumatic third nerve injury may be partial or complete. The patient may complain of oblique diplopia and blurring near vision as a result

of decreased accomodation. Varying degrees of ptosis in combination with impaired adduction, elevation and depression is noted on examination. With pupil involvement, there is efferent pupillary defect. Over time abberant regeneration may occur in the form of pseudo Von Grafe, pseudo Duane, pseudo Argyl Robertson Pupil.

Management- 1/3 rd spontaneously recover within 6-12 months. Residual ptosis and ophthalmoparesis are managed surgically.

E. Trochlear nerve injury

The fourth nerve is vulnerable to trauma due to following reasons:

a. longest cranial nerve

b. slender

c. exits dorsally from brain

The most common site of trochlear nerve injury is the portion traversing the subarachnoid space. Contrecoup motion of brainstem during trauma results in contusion of one or both trochlear nerves by the tentorial incisura. Patients usually complain of vertical binocular diplopia that increases on down gaze. Patient may also have a head tilt to the side opposite the paresis. The Bielschowsky head tilt test is useful in localizing dysfunction to the superior oblique muscle – ipsilateral hypertropia worsening on opposite gaze and same side head tilt

The prognosis of functional recovery in fourth nerve palsy is relatively good. Approximately 40% of patient may recover within 8 months after injury. If sufficient recovery has not occurred by 6 to 12 months and base up prism prove unsatisfactory, then surgical correction is considered.

F. Abducent nerve injury

It occurs in severe head injury with loss of consciousness.

a.The most common site of injury to abducent nerve is where it passes beneath the petroclinoid ligament to pierce the dura on entering the posterior cavernous sinus.

b. Next common site is in the cavernous sinus. There will be associated other cranial nerve palsies

c. Trauma to tectum of pons can result in damage to the abducent nucleus. Because the genu of fascial nerve loops over abducens nucleus, there may be concomitant facial nerve palsy.

d.Increased intracranial pressure from posttraumatic hydrocephalus or cerebral edema may compromise the abducens nerve as it traverses the subarachnoid space. Thus it can be a false localizing sign[14].

Approximately 40 – 50% of patients with traumatic sixth nerve paresis spontaneously improve within 6 – 12 months. Surgery may be considered if recovery is incomplete after 6 – 12 months and the patient is symptomatic even after conservative measures

G. Central disorders of ocular motility:

Trauma to internuclear and supranuclear pathways of the ocular motor system in the brainstem and cerebrum may be manifested by a variety of motility disturbances. The features of ocular motility disturbances may be:

- Internuclear ophthalmoplegia occurring in trauma to mescencephalic region- MLF
- Skew deviation occuring in disruption of vestibular input
- Horizontal gaze palsy occuring in lesions at pontine level
- Vertical gaze palsy occur in damage to rostral interstitial nucleus of the MLF
- Convergence spasm or insufficiency, Impaired accommodation, Disorders of fusion, Acquired esotropia occuring as a part of dorsal midbrain syndrome

H. Facial nerve injury

Trauma to facial nerve with subsequent impairment of orbicularis oculi function and reflex lacrimation may lead to exposure keratopathy or corneal blindness.

Sites of facial nerve injury:

a. Facial nerve nucleus or fascicles- in brainstem injury

b. Intracranial portion of facial nerve in fallopian canal- in temporal bone fracture

c. Extracranial portion of facial nerve- in blunt or penetrating injuries

Ocular manifestations of facial nerve palsy:

- Lagophthalmos
- Exposure keratitis
- Keratoconjunctivitis sicca
- Impaired blink
- Lower lid retraction
- Paralytic ectropion
- Epiphora
- Brow ptosis
- Abberant regeneration

Management:

- Exposure keratitis- topical lubricants and lid tapping
- Tarsorraphy- if conservative measures are insufficient
- Use of gold weights for upper lid in combination with surgical correction for lower lid laxity provides long term functional and cosmetic results

D. ORBITAL AND ADNEXAL INJURIES

A. Periocular lacerations :

Careful inspection of periocular lacerations provides abundant clues to the nature and extent of the injury. The general considerations

include the location and depth of injury, tissue loss, impaired function and presence of foreign body.

a.Marginal lid laceration:

Lacerations lateral to puntum- unlikely to involve canaliculus

Lacerations medial to punctum- likely to be associated with canalicular laceration

b.Non marginal laceration:

Superficial- involving skin and orbicularis

Deep - 1. upper eyelid involves septum, levator aponeurosis and muscle, lacrimal gland, trochlea and superficial neurovascular bundles.

2. lower eyelid the septum and inferior oblique muscle may be injured.

3. medial canthus: canaliculus, lacrimal sac, medial canthal tendon

4. lateral canthus: lateral retinaculum, facial nerve may be injured.

Complex eyelid lacerations may present with large gaping wounds. As the intact portion of eyelids contract, large gaps develop in the eyelid contour giving the appearance of an avulsion. In these cases of

pseudo- tissue loss, primary repair is possible. However in case of tissue loss, an effort should be made to save tissue if available.

In penetrating injuries to upper eyelid, it is important to assess levator function preoperatively. In the setting of acute soft tissue edema, the levator function is often underestimated. But a careful examination can help to distinguish a direct levator injury from a traumatic third cranial nerve injury.

It is important to have a high index of suspicion for the presence of foreign body. Based on the history and initial inspection of the patient, the appropriate imaging modality help to determine the location, depth and possibly the nature of foreign body prior to commencing surgical repair.

Treatment of periocular injuries:

Acute treatment: Wounds should be thoroughly cleaned. Adequate debridement of particulate matter prevents the late sequelae of dermal tattooing.

Timing of repair:

In cases of isolated periocular trauma, timing of repair is not critical. More emergent intervention needed in case of associated globe rupture. In most cases, a delay of 12- 36 hrs from time of injury doesn't alter surgery results.

Laceration repair:

Superficial non marginal injury repair- can be repaired primarily without difficulty. Marginal full thickness injury – primary objective is to restore the local anatomy with appropriate alignment of the eyelid margin, tarsus and skin. Eyelid alignment is best achieved by using the eyelash line, gray line and meibomian units as local landmarks. These structures can be adequately positioned with two to three 6-0 or 7-0 sutures placed along eyelid margin.

c. Levator lacerations:

If a levator muscle complex injury is suspected, it is important to explore the muscle and aponeurosis through an upper eyelid crease incision. If the aponeurosis is detached from the tarsus, repair is directed at suturing the cut end of the aponeurosis to the upper anterior face of tarsus with either silk or polyglactin suture.

d. Lateral canthus injury:

If the attachment of lateral canthal tendon is disrupted, repair should be directed posteriorly using a permanent suture anchored to the periosteum. If there is no periosteum, the tendon can be fixed to the orbital rim through appropriately drilled holes.

e. Medial canthal injury:

Lacerations extensive enough to lacerate the medial canthal tendon are also associated with canalicular injuries. The medial canthal tendon is optimally repaired with a permanent suture fixed to the periosteum

At the level of posterior lacrimal crest, if the bone or periosteum is not intact, transnasal wiring of medial canthus is the possible solution.

f. Canalicular injury:

In the majority of cases, there is usually no doubt that the canalicular system is disrupted based on disruption of wound. In cases that are not readily apparent, probing of the canalicular system and inspection of wound for direct visibility of the probe help establish the diagnosis. In cases where depth of wound is not seen, canalicular information may provide additional information.

The general concept of canalicular repair is to pass an internal stent to bridge the laceration and to reestablish continuity of the disrupted canalicular system. Stents can be monocanalicular – Viers rod, Silastic tube or bicanalicular- pigtail, nasolacrimal probe.

g. Tissue loss:

(i) Superficial avulsion:

In cases of tissue loss anterior to the septum, primary granulation is considered. When allowed to granulate, these wounds require more attention than primary repair, as the epithelialisation is prolonged and there is increases risk of infection.

(ii) Deep tissue loss:

Large confluent loss of the anterior lamella of the eyelids including skin and muscle, which exposes underlying fat is not suitable for granulation. These wounds should be covered with full-thickness skin grafts or musculocutaneous flaps in acute setting.

B. Injuries to orbit:

Based on the patterns of fracture, the orbit is divided into four zones:

1. supraorbital area of the frontal bone
2. nasoethmoid area
3. zygoma
4. internal orbit

Thin section CT imaging is the gold standard in diagnosis of orbital injuries. CT scan not only provide precise documentation of skull features, but also gives the information about the soft tissues of the globe, orbit and cranial vault.

Type of fracture	Associated soft tissue injury	Frequency	X-ray view	CT finding	Clinical signs	Management
Supra orbital frontal fractuies	Forehead contusion or laceration	Less common	Antero posterior view	Fracture of supra orbital frontal bone	Ptosis, proptosis	Fractured segments replaced anatomica held with wires
Nasoethmoorbital fractures	Edema over medial canthal region	Common	AP view – fracture of nasal bone, fracture of the junction of the frontal process of maxilla with frontal bone, fracture of medial orbit, fracture of infra orbital rim	Same as x-ray	Nasal flattening with loss of dorsal height, increased angle between lip and collumella	Reduction and fixation of all fractured segments May need acute bo grafting.
Zygoma fracture	Periorbital and subconjuctival ecchymosis	More common	Water's view fracture through inferior orbital rim Caldwell view displacement of zygomatico frontal articulation	CT precisely define displacement	Swelling of cheek and face with contour irregularities, loss of malar prominence, step deformity at the zyomaticomaxillr ay articulation	Open reduction an fixation with miniature metal plates

Continued:

Type of fracture	Associated soft tissue injury	Frequency	X-ray view	CT finding	Clinical signs
Internal orbital fractures: (i) orbital apex fractures	Periorbital contusion	Less common		CT through orbital apex – fracture at or adjacent to optic canal	CSF leaks, traumatic optic neuropathy, carotid cavernous fistula
(ii) orbital roof fracture	Periorbital contusion, lid laceration	Common in young children	Anteroposterior view show fracture	Same as X-ray	CSF rhinorrhea, pneumocephalus, sub periosteal haematoma, ptosis
(iii) medial orbital wall fracture	Depressed nasal bridge, traumatic telecanthus	Less common	Anteroposterior view show fracture	Fracture involving frontal process of maxilla, lacrimal bone, ethmoid bone along medial wall or orbit	Epistaxis, CSF rhinorrhoea, orbital haematoma, damage to lacrimal drainage system

(iv) Orbital floor fractures :

It can be: Direct fractures- involves orbital rim called impure fracture

Indirect fractures (blow out)- don't involve orbital rim

An orbital blow out fracture should be suspected in any patient who received periorbital blow forceful enough to cause ecchymosis.

Physical examination reveals

1. Eyelid signs- ecchymosis, edema
2. Diplopia with limitation of upgaze, downgaze or both- due to entrapment of the inferior rectus or its adjacent septa in the fracture
3. Enophthalmos and ptosis of the globe- These occur with large fractures in which the orbital soft tissues prolapse into the maxillary sinus.
4. Hypoesthesia in the distribution of the infraorbital nerve
5. Emphysema of the orbit and eyelids

Xray anteroposterior view of orbit and facial bone shows tear drop sign

CT scans with coronal or saggital views are usually indicated to guide treatment. The majority of blowout fractures don't require immediate

intervention. They are observed for 7-10 days so that swelling and orbital haemorrhage can subside. Blowout fracture in paediatric patients, where attempted ocular excursions can stimulate the oculocardiac reflex leading to bradycardia, nausea and pain, immediate intervention is needed.

Indications for surgical intervention are:

1. Diplopia with limitation of upgaze or downgaze within 30 degrees of primary position, positive traction test 7-10 days after injury and with radiological confirmation of fracture of orbital floor.
2. Enophtholmos that exceeds 2mm and that is cosmetically unacceptable to the patient
3. Large fractures involving at least half of the orbital floor, particularly when associated with large medial wall fractures

Surgery is best done within two weeks for easy manipulation of the scar tissue and fibrosis of entrapped tissue. The surgical correction can be made through an infraciliary approach or conjunctival incision combined with lateral cantholysis. The steps are,

(i) elevation of periorbita from orbital floor

(ii) release of prolapsed tissue from the fracture

(iii) placement of an implant over the fracture to prevent recurrent adhesions Complications of blowout surgery include, decreased visual

acuity or blindness, diplopia, undercorrection or overcorrection of enophthalmos, lower eyelid retraction, infraorbital nerve hypoesthesia, infection, extrusion of implant, lymphedema and damage to lacrimal pump.

REVIEW OF LITERATURE

Ocular trauma due to mechanical injuries is one of the major cause of visual impairment world wide. There are in excess of 2 million cases of ocular trauma per year in the world and of these about 40,000 cases sustain severe visual impairment. The incidence of ocular trauma are increasing due to modernization and increased industrialization. So clinical study of ocular morbidity due to mechanical injuries in patients attending the tertiary hospital is done in this thesis. This study includes history, clinical examination, investigations and the morbidity is assessed qualitatively and quantitatively.

Ocular injuries are classified anatomically into anterior segment, posterior segment, neuroophthalmic injuries and orbital and adnexal injuries. The nature of mechanical injury, occupation of the

individual, duration between injury and examination, initial visual acuity and final visual acuity are studies.

A retrospective study on epidermiology of adult eye injuries by Ksenijia Karaman et al states that initial visual acuity predicted the visual outcome. The severity of ocular injury was independent of the age of the individual. Penetrating injury to eye leads to more ocular morbidity than blunt injury. Similar results were found by De Juan et al.

A prospective study on the profile of ocular trauma at tertiary eye care by D.V.Singh et al revealed that males were injured more (88.5%). One third of injuries occurred in pediatric age group. This is supported by the retrospective study in a tertiary eye care in Eastern India by Dr.Sucheta Parija et al, who had found similar ratio of involvement among males and females. Right eye involved in 50.1%, left eye in 46.8% and both eyes involved in 3.1%. Facial injury associated with ocular injury was seen in 20.1% of eyes. 3% of patients were able to reach tertiary centre within 48 hours.

A prospective study on expected effect of treatment on the ratio of visual deficiency after ocular trauma by Virgilio Lima-Gomez et al showed that the most frequent feature of poor visual prognosis is afferent papillary defect and globe rupture. A retrospective study on pediatric ocular trauma by M.R.Shoja et al states that, high frequently

of ocular injuries occur at streets and roads. A major group of children belong to poor socioeconomic class. In this study penetrating injuries predominate over blunt injuries. This may be due to the fact that ocular injuries treated as out patient injuries were not included.

A retrospective study by Mohammed Yashir Arafat et al on injuries to eye has stated that, the incidence of ocular trauma in males are 84% and in females are 16% which is comparable to the study by D.V.Singh et al. The study also had found that right eye is involved in 66% and left eye in 34%. The post treatment visual acuity between 6/6 and 6/60 was 74%.

Atkari et al in this study had found that males are move involved 55.8% and females 44.2%. This is comparable to other studies. In his study he had noted left eye involvement (59.9%) more than right eye (42.7%). Bilateral involvement is rare (5.6%). This is in contrast to other studies, where right eye involvement outnumbered left eye. The common age group involved was 16-35 years, which is the active period of life. Most of them were farmers (32.1%) and house wives (26.2%). His study also shows that only 43.3% came for treatment within 7 days and very minimal of 3.09% reported for treatment within 6 hours.

The visual outcome of perforating globe injuries depends on the type of injury sustained. Injuries due to sharp objects cause localized damage and visual prognosis is better. Blunt injuries usually cause globe disorganization and so visual prognosis is poor. Enucleation was performed previously and now it has declined because of better understanding of pathophysiology, improved surgical techniques and antibiotic use to prevent sepsis.

MATERIALS AND METHODS

The prospective study was conducted on 100 cases of ocular trauma attending Tirunelveli Medical College Hospital. Patients with ocular injury attending ophthalmology outpatient department were randomly included in the study.

Inclusion criteria were:

- Patients of all ages

- Both males and females
- All co-operative patients
- Irrespective of economic status

Exclusion criteria:

- Unconscious patients
- Patients not co-operative for examination and procedures
- Terminally ill patients

A detailed patient data including age, sex, occupation, residential area, nature of injury like road traffic accident, assault, accidental fall, accidental injuries due to stone, needle pricks etc.., were taken. The presenting complaints, time interval between injury and examination, directly came for treatment or referred from peripheral hospital and initial medical assistance taken were also recorded.

Patients unaided visual acuity at the time of presentation was recorded using Snellen's chart. Orbit, eyelid and adnexa were examined by diffuse illumination. Lid injuries, periorbital contusion and injuries around orbit were recorded. Slit-lamp examination was done to patients with trauma attending our out patient department and also to trauma patients of emergency department who needed slit lamp examination.

Conjunctiva examined for any injury like tear and sub-conjunctival haemorrhage. Cornea examined for abrasion, tear, foreign body, extent and depth of injury. Anterior chamber reaction in the form of flare and cells were noted and recorded. Iris examined for sphinter tear and iridodialysis. Pupil examination done to note direct and indirect light reflexes, mydriasis, relative afferent papillary defect and pupil distortions. Lens examination done to look for any penetrating injury, traumatic cataract, subluxation and dislocation.

Posterior segment examination done by direct ophthalmoscopy in all patients provided the anterior segment did’nt preclude fundus examination. +90D examination and indirect ophthalmoscopy done for needed patients. Optic disc, macula and retina were examined for disc edema, macular hole or haemorrhage, retinal haemorrhage, tear, detachment and infarcts.

Intraocular pressure recorded by Applanation tonometry or non contact tonometry in trauma patients attending our department and Schiotz tonometry done to others.

B scan done to assess the posterior segment in patients with blunt and penetrating injury to eye ball and in those patients for whom fundus examination cant be done due to hazy media. CT scan facial bones and X- ray orbit done to needed patients. Final diagnosis was made. Treatment given to these patients either medically or surgically

depending on the case. Final visual acuity on discharge was recorded. Causes for poor visual recovery in the patients included in the study were noted. All the datas are included in a proforma and analysed. All medico-legal cases are duly entered in accident register.

OBSERVATION

STATISTICAL ANALYSIS

The study subjects were analyzed and interpreted in terms of their demographic characteristics, type of injury, type of morbidity, eye/ eyes involved in the injury and visual outcome. The continuous variables were interpreted by Students' 't' test and categorical variables were interpreted by χ^2 (Chi-square) test where ever applicable. The above statistical procedures were performed by IBM SPSS statistics 20. The P- values less than 0.05 ($P<0.05$) were defined as statistically significant in two-tailed test.

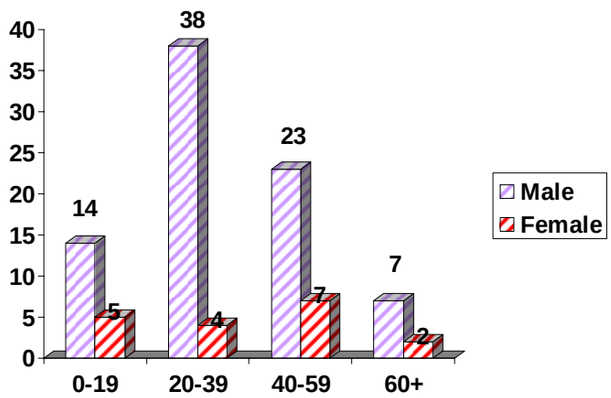
RESULTS AND OBSERVATION

The different type of injury in the persons included in the study were classified according to their age and sex.

Table-1.Classification based on age with sex.

Age (years)	Male		Female		Total	
	No	%	No	%	No	%
0-19	14	17.1	5	27.8	19	19.0
20-39	38	46.3	4	22.2	42	42.0
40-59	23	28.1	7	38.8	30	30.0
60+	7	8.5	2	11.1	9	9.0
Total	82	100.0	18	100.0	100	100.0
Mean ± SD	34.9±16.0		35.7±18.2		35.1±16.3	
Range	8-80		8-65		8-80	
't'	0.186				-	
Significance	P>0.05					

Table.1. Classification based on age & sex

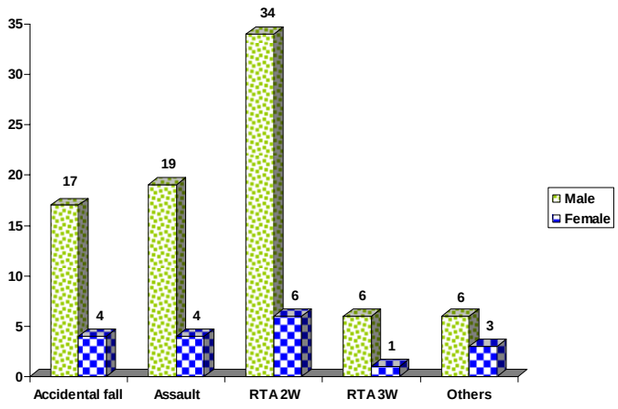


The above table-1 shows the percentage distribution in the study subjects according to their sex with age. The mean age of males were 34.9±16.0 and females were 35.7±18.2 (years). They were not significantly differed in respect of their age (P>0.05). The mean age of the total subjects was 35.1±16.3 years with range 8-80 years. The Males were 82% and females were 18%.

Table-2. Sex wise distribution of accidents.

Accident	Gender			χ^2	Df	Sig.
	Male	Female	Total			
Accidental Fall	17	4	21	6.785	5	P>0.05
Assault	19	4	23			
RTA 2W	34	6	40			
RTA 4W	6	1	7			
Others	6	3	9			
Total	82	18	100			

Table.2. Sex wise distribution of accidents

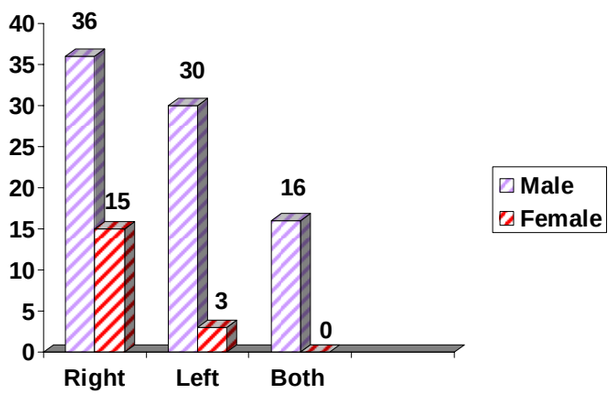


The sex wise accidents were shown in the above table -2. Among the injuries, road traffic accidents were 47% , assault – 23%, accidental fall – 21%. In the road traffic accidents, accidents by 2 wheeler was 85% . The results revealed that there was no significant relationship between the gender with accidents ($P>0.05$).

Table-3. Sex wise distribution of affected eyes.

Eyes	Gender			χ^2	Df	Sig.
	Male	Female	Total			
Right	36	15	51	9.787	2	P<0.01
Left	30	3	33			
Both	16	0	16			
Total	82	18	100			

Table.3. Sex wise distribution of affected eyes



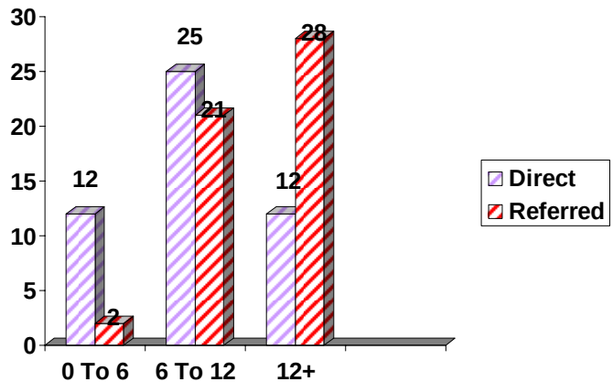
The above table-3 associates sex of patients with eyes affected. The females of 83.3% (15) were affected with right eyes and 43.9% (36) of males were affected with right eyes. Similarly, 19.5% (16) of males only were affected with both eyes. The above

associated eyes with gender was statistically significant (P<0.01).

Table-4.Time of examination since admission.

Time of examination (hrs)	Type of admission			χ^2	Df	Sig.
	Direct	Referred	Total			
0-6	12	2	14	13.856	2	P<0.001
6-12	25	21	46			
12+	12	28	40			
Total	49	51	100			

Table.4. Time of examination since admission



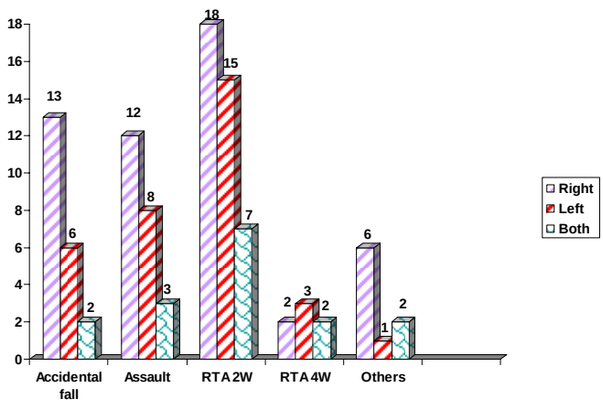
The admission with duration was related in the above table -4. The direct admission was examined early compared to referred cases. These referred cases has got initial medical attention in the peripheral

hospitals.

Table-5. Laterality of injury.

Injury	Eyes				χ^2	Df	Sig.
	Right	Left	Both	Total			
Accidental fall	13	6	2	21	6.690	10	P>0.05
Assault	12	8	3	23			
RTA 2W	18	15	7	40			
RTA 4W	2	3	2	7			
Others	6	1	2	9			
Total	51	33	16	100			

Table.5. Laterality of injury

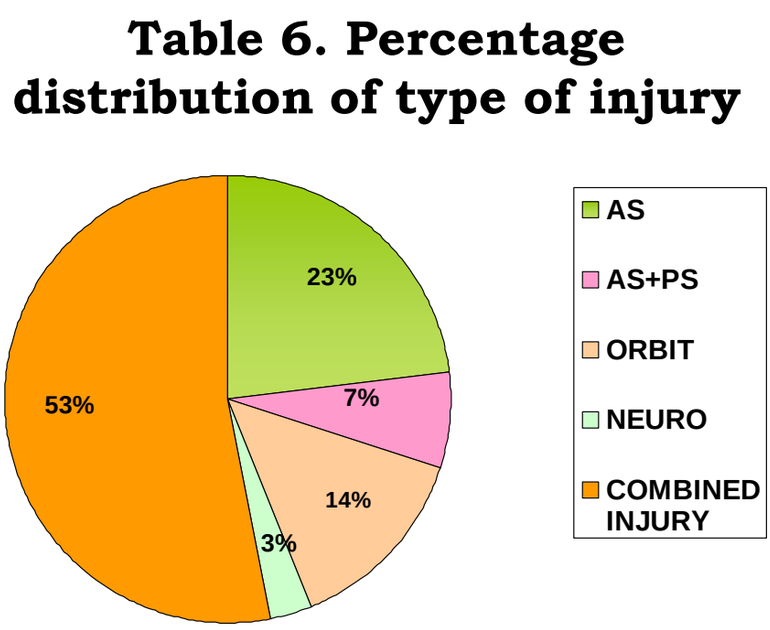


The accidents wise affected eyes were shown in the above table -5.

The results revealed that there was no significant relationship between the accidents with affected eyes (P>0.05).

Table-6. Percentage distribution of type of injury.

Type of Injury	No	%
AS	23	23%
AS+PS	7	7%
ORBIT	14	14%
Neuro	3	3%
Combined injury	53	53%
Total	100	100%



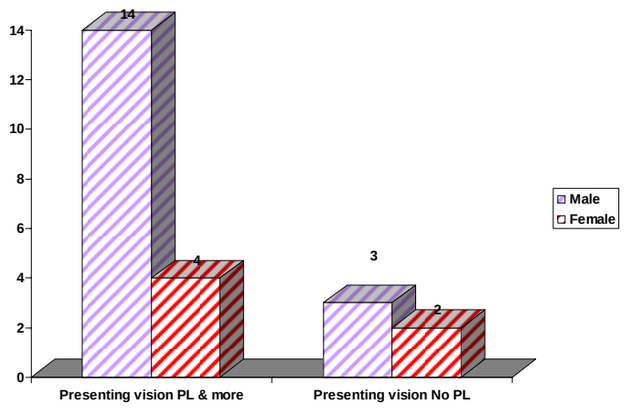
The above table -6 describes the Combination of type of injury. This table shows that combined injury involving more than one

segment accounts to 53%. Anterior segment injury alone is next commonly involved.

Table-7. Visual outcome in Traumatic optic neuropathy

Sex	Presenting vision PL and more	Presenting vision No PL	Improved visual outcome
Males	14	3	6
Females	4	2	1
Total	18	5	7

Table.7 Visual outcome in Traumatic optic neuropathy

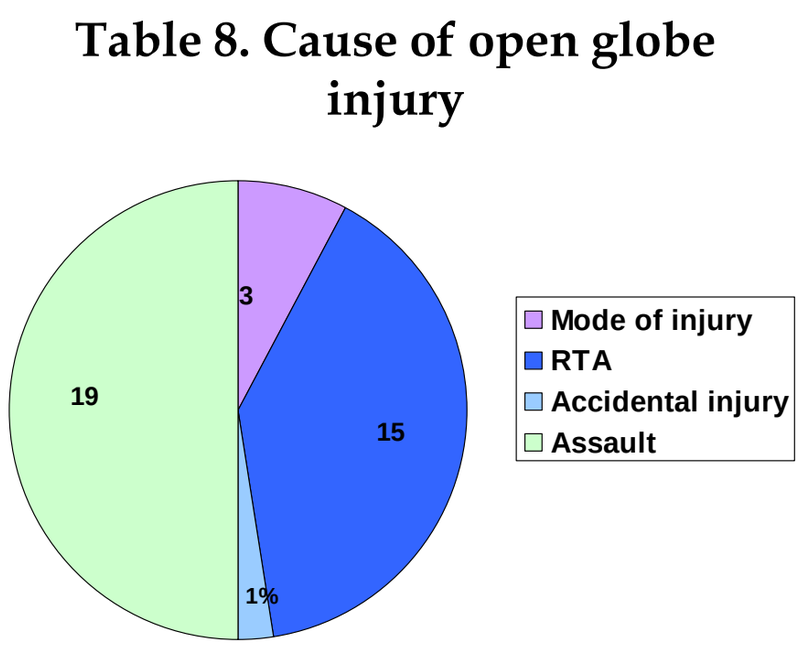


This table shows 18 patients had traumatic optic neuropathy. Initial visual acuity of PL and above were 18, and no PL at

presentation were 5. Of these 7 patients showed improvement with treatment. All these 7 patients had PL and above visual acuity at presentation.

Table- 8: Cause of open globe injury:

Mode of injury	No of persons
RTA	3
Accidental injury	15
Assault	1
Total	19



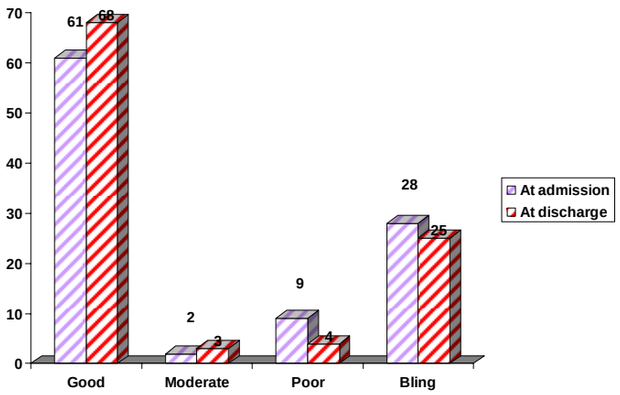
Open globe injury noted in 19 patients. Common cause for open globe

injury was accidental injury with sharp objects. Of these 6 patients developed endophthalmitis. Two of them had intraocular foreign body.

Table-9. Category of vision at different intervals.

Vision	At admission	At discharge	At 1 month follow up
	Persons	Persons	Persons
Good	61	68	69
Moderate	2	3	2
Poor	9	4	5
Blind	28	25	24
Total	100	100	100

Table.9. Category of vision at different intervals



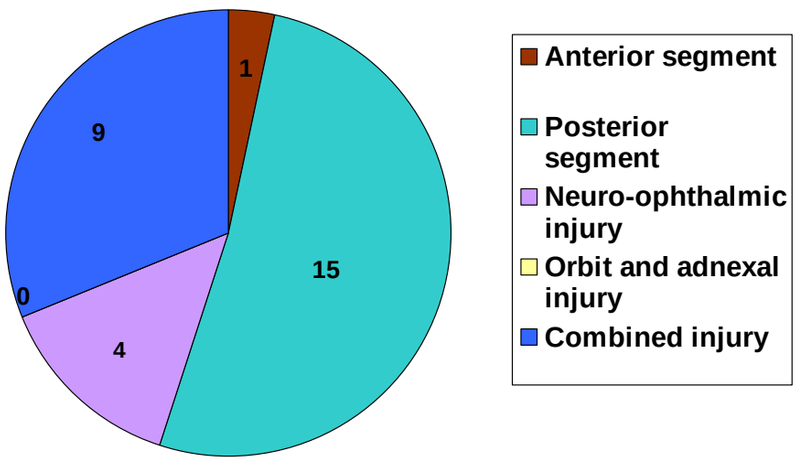
Visual acuity categorised as Good vision- 6/12-6/6, moderate vision – 6/36-6/24, poor vision – 3/60-6/60 and blind – no PL – 3/60. This table shows injured eyes with good vision at presentation improved. Of the patients with blind eyes as per WHO definition(i.e) <3/60 , did' nt show much improvement.

Table-10: Analysis of patients with poor visual outcome:

Nature of injury	Poor visual outcome at discharge <6/60
Anterior segment	1
Posterior segment	15
Neuro-ophthalmic injury	4

Orbit and adnexal injury	-
Combined injury	9
Total	29

Table.10. Analysis of patients with poor visual outcome



The cause for visual acuity of < 6/60 is shown in this table. Posterior segment injury showed poor visual outcome in 15 cases. Orbit and adnexal injury and anterior segment injury alone had good visual outcome.

DISCUSSION

Ocular injuries are one of the common cause of ocular morbidity nowadays. It has been estimated that, 1.6 million people are blind in the world due to ocular trauma, 2.3 million people are visually impaired due to trauma and 19 million people are unilaterally blind due to ocular trauma. Males are more affected than females and ocular injuries are common in younger age groups.

In this study, common age group involved was between 20 – 39 years 42%. This is comparable to study by Atkari et al[17]. This age group is the economically productive age group and ocular injury to this age group cause a great deal of economic loss to the country. Although students (22%) formed the major subgroup, drivers (17%) , coolies (22 %)and farmers (14%) were also involved. But study by Atkari et al showed most of the involved patients were farmers (32.1%). High incidence in students may be due to their increased involvement in risk activities. They play hazardous and dangerous games. This could be the reason for high incidence in students.

Males are more commonly affected (82%) compared to females of (18%.) Male predominance and peak age involvement in our study were similar to other studies. This is due to the fact that, males are more involved in outdoor activities. A prospective study on the profile of ocular trauma at tertiary eye care by D.V.Singh et al[18] showed similar results. This is also supported by the retrospective study in a tertiary eye care in Eastern India by Dr. Sucheta Parija et al[19] . This is comparable to the demographic profile noted in a study in South India by Pieramici DJ, Sternberg P Jr, Aaberg TM et al[20] . Involvement of males more than females, who are the bread winner for most of the families will have an impact on productivity of our nation for sure.

The proportion of right eye involvement (51%) was more than left eye (33%), which is in agreement with other studies. Similar results were noted in a study by D.V.Singh et al.

In our study among the mode of trauma, road traffic accident(47%) was the major cause for mechanical injuries to eye. Assault was the cause for 33%, accidental falls for 21% . Of the road traffic accidents, two wheeler accidents were responsible for 40%. Similar results were seen in De Juan E et al[21] study on penetrating ocular injuries. This may be due to increased vehicular traffic and increased population. Rash driving is the major reason for two wheeler accidents.

In our study, 49% of patients presented directly to our hospital and 51% had initial medical assistance and referred from peripheral hospitals. Of the directly presented patients, 12 patients have come after 12 hours. In our study 14% have reported within 6 hours of injury. But in Atkari et al study only 3.09% patients have reported within 6 hours. Now due to increased communication and transport facilities, more patients are able to reach tertiary care hospitals earlier. Initial hours after injury are important as early treatment helps in speedy recovery. We noted that patients in whom treatment was initiated within 12 hours had better visual acuity.

Based on injuries sustained, anterior segment alone was involved in 23%, anterior and posterior segment in 7%, neuro-ophthalmic manifestations alone in 3%, orbital and adnexal injury alone in 14% and combined injuries in 53%. Anterior segment and orbital injuries alone had better visual outcome and less morbidity than neuro-ophthal, posterior segment and combined injuries. This was in agreement with study by Virgilio Lima Gomey [22]

In our study, 18 patients had traumatic optic neuropathy. Of these 7 patients (39%)showed improvement with steroid treatment. This is comparatively less than other study by J.A.Mauriello et al[23], which showed 56.25% with steroid treatment. This difference may be due to delayed presentation and so delay in initiation of steroid treatment. We could find that patients with at least PL vision on presentation improved with treatment. Of the 7 patients who showed visual improvement, all had vision of PL and above.

In our study, open globe injury was found in 19% of patients and in them 6 patients (30.1%) developed endophthalmitis. Similar incidence was noted in a study by D.V.Singh et al. This is comparatively high compared to study by Thompson SW, Rubsamen PE et al[24] . The delayed presentation to the tertiary centre may be the reason for this high occurrence. In our study we had two patients with open globe injury , who subsequently developed endophthalmitis after foreign body removal. This was due to late presentation to our hospital, after 48 hours. Lens capsule injury is associated with endophthalmitis. This is seen in one patient, in our study. This is also noted in studies by Reynolds DS et al[25] and Schmidseder E, et al[26]. The presence of intraocular foreign body, lens capsule injry and delayed presentation are risk factors for developing endophthalmitis.

In our study, 71% had good vision at discharge and one month follow up. Patients who presented with good visual acuity had good visual outcome. This is also shown in study by Sternberg P Jr et al and also by Ksenija Karaman et al[27] . The presenting visual acuity is one of the important prognostic factors for good visual outcome.

Poor visual outcome of <6/60 were seen in 29 % in our study. The major cause for this is found to be posterior segment injury alone (51%). Combined injury accounted for (31%). Poor visual outcome in posterior segment injury is due to high incidence of vitreous disturbance, incarceration of retinal tissue, vitreous haemorrhage and associated retinal and choroids detachment. This is in agreement with study by Cleary PE, Ryan SJ et al[28] .

CONCLUSION

Ocular injury due to mechanical trauma can cause morphological and functional impairment to ocular structures and vision.

- In our study, younger age group between 20 – 39 years are commonly involved in ocular injury due to mechanical trauma. Students are more involved in mechanical injuries. This age group should be focused and made aware of preventive aspects against mechanical injury. Children should be guarded from using sharp objects. Children should be allowed to play under parent supervision.
- Males are more involved than females in our study. This is due to males being involved in outdoor activities for occupation. So, to prevent injuries during occupation wearing of protective eye shields while working should be encouraged.
- While considering the mode of injury, road traffic accidents were responsible for ocular injury in many patients. Of the road traffic accidents, two wheelers predominated. To prevent this, people should be made aware of wearing helmets which

could reduce ocular injuries to a great extent. To reduce injuries due to four wheeler accidents, wearing of seat belts should be given priority and should be enforced by law.

- Early institution of treatment, once diagnosis is made, reduced ocular morbidity in our study. So people should be made aware of seeking early medical help. This can be achieved by mass media to disseminate ocular trauma related information.
- Posterior segment injury and open globe injury had poor visual outcome. These patients should be referred to retina surgeon and tertiary referral centre at the earliest. So primary ophthalmologists should diagnose this earlier and refer to tertiary hospitals where such facilities are available. protective measures should be taken in working places and
- It is necessary to implement preventive measures to prevent mechanical trauma to eye. Wearing seat belts and helmets should be made compulsory. Legislation alone is not successful for this effort. Education regarding safety measures and measures to be taken if injury occurred should be done through mass media. These measures will reduce ocular morbidity due to mechanical trauma to a great extent.

PROFORMA

Serial No:

Hospital No: MLC / Non MLC Date:

Name: Age: Sex:

Occupation and income: Date of admission:

Date of surgery:

Date of discharge:

Address:

Complaints: Duration between trauma and examination:

0 to 6hrs / 6 to 12hrs / 12 to 24hrs /

days/weeks

Hospital details:

RTA/assault/other injuries

Right eye/left eye/both eyes

Loss of consciousness – yes/no

Painful/painless

Visual disturbance – yes/no

Movements of eye – possible/restricted

Diplopia – yes/no

Watering of eye – yes/no

Photophobia – yes/no

Head ache – yes/no

Vomiting – yes/no

Swelling

ENT symptoms – bleeding nose /tinnitus

CLINICAL EXAMINATION:

Right eye Left eye

EOM

Anterior segment examination:

(SLE)

Extra ocular movements

Lid

Conjunctiva

Cornea

Limbus

Anterior chamber

Iris

Pupils

Lens

Duct

Tension

Posterior segment examination

Fundus – Media

Disc margins

C : D ratio

Vessels

Haemorrhages

Macula

Periorbital region

Swelling /ecchymosis/emphysema

Orbital margins

Infra orbital anesthesia

Vision

Colour vision

Field of vision

Others

ENT examination

Central nervous system examination:

Conscious/unconscious

Oriented / disoriented

Memory

Speech

Cranial nerve examination

Motor system

Sensory system

Speciality opinion:

Neuro surgery/neuro medicine/plastic surgery/orthopedics

Investigations:

X-ray skull

CT scan Brain and Orbit

MRI scan brain and Orbit

Others

Final clinical diagnosis:

Treatment: Medical/surgical/others

Follow up visits:

Statistical analysis

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KEY WORDS TO MASTER CHART

RTA – Road traffic accident

RE – Right eye

LE – Left eye

BE – Both eyes

AS injury – Anterior segment injury

PS injury – Posterior segment injury

Neuro – Neuro ophthalmic injury

VA on A – Visual acuity on admission

VA on D – Visual acuity on discharge

SCH – sub conjunctival haemorrhage

 # - Fracture

EL – Eye lid

AC – Anterior chamber

HM – Hand movements

PL – Perception of light

NO PL – No perception of light

MC – Mature cataract

FTCT – Full thickness corneal tear

RD – Retinal detachment

CD – Choroidal detachment

IOFB – Intraocular foreign body

VH – Vitreous haemorrhage

IViAB – Intravitreal antibiotics

RCS – Retina choroids sclera

K tear – Corneal tear

PCIOL – Posterior chamber intraocular lens

P&B - Pad and bandage

EDH – Extra dural haemorrhage

SDH – Sub dural haemorrhage

 # BB – Fracture both bones

S.NO	NAME	AGE	SEX	RE	LE	BE	TYPE OF INJURY	DIRECT	REFERRAL	0to 6	6 to 12
1	Subramanian	50	M			Y	accidental fall				
2	Abuthakir	18	M		y		RTA 2 wheeler	y			y
3	Sangeetha	8	F	y			RTA 2 wheeler		y		y
4	Kuttiraj	48	M	y	y		RTA 2 wheeler	y			y
5	Kalliappan	44	M		y		assault	y			y
6	Vinoth	26	M	y			RTA 2 wheeler	y		y	
7	Thangavel	57	M		y		RTA 2 wheeler	y		y	
8	Muthukrishnan	47	M		y		RTA 2 wheeler	y			
9	Mytheen pitchai	28	M		y		RTA 2 wheeler		y		
10	Mohammed nasar	40	m	y			assault		y		Y
11	Jeyaraman	27	M		y		RTA 4 wheeler	y		y	
12	Ganesan	20	M	y			RTA 2 wheeler		y		
13	Chelliah	57	M		y		accidental fall		y		y
14	Shunmugathai	43	F	y			accidental fall RTA	y		y	
15	Gandhi	60	M			y	accidental fall		y		
16	Packiaraj	42	M	y			assault	y			y
17	Karupasamy	36	M	y			thorn prick	y			

18	Krishnan	80	M			y	assault	y			
19	Muthuraj	18	M	y			RTA 2 wheeler	y			
20	Balasubramanian	40	M			y	RTA 2 wheeler	y			
21	Ravi	18	M			y	RTA 2 wheeler		y		
22	Kothaiammal	65	F	y			acci hit in a iron pipe	y			y
23	Banumathi	40	F	y			accidental fall	y			
24	Subbiah	66	M	y			hit by goat horn		y		
25	Kumar	22	M		y		accidental fall	y			y
26	Kalleswari	37	F	y			RTA 3 wheeler	y		y	
27	Santhan	37	M		y		RTA 2 wheeler	y			
28	Chidambaram	35	M	y			assault		y		
29	Karthikeyan	38	M	y			RTA 2 wheeler	y		y	
30	Sollamuthu	29	M	y			hit by goat horn		y		
31	Aaron	47	M			y	assault stamped in chest	y			y
32	Mariraj	32	M			y	RTA 2 wheeler		y		
33	Sundari	56	F	y			RTA 2 wheeler		y		
34	Bagyanathan	37	M		y		assault	y			y
35	Ramakrishnan	35	M	y			RTA 2 wheeler		y		
36	Guna	23	M		y		RTA 2 wheeler		y		
37	Vinoth kumar	15	M	y			RTA 2 wheeler		y		y

38	Kannan	23	M	y			RTA 2 wheeler	y			y
39	Manikandan	25	M		y		RTA 4 wheeler		y		y
40	Gomathi	16	F	y			RTA 4 wheeler		y		
41	Malaiarasan	19	M	y			RTA 2 wheeler		y		
42	Athilingam	65	M	y			assault with stick		y		y
43	Sudali	21	F	y			RTA 2 wheeler	y			y
44	Saroja	35	F	y			assault		y		y
45	Alwar	55	M	y			assault hit by stay wire	y		y	
46	Yesudass pandidurai	28	M	y			RTA 2 wheeler			y	
47	Sankaranarayana n	56	M	y			assault	y		y	
48	Muthammal	61	F	y			assault		y		y
49	Sundaram	55	F		y		RTA 2 wheeler		y		y
50	Ramesh	25	M	y			RTA 4 wheeler		y		y
51	Summons	51	M	y			assault		y		y
52	Saravanan	40	M			y	RTA 2 wheeler		y		y
53	Pathirakali	28	F	y			assault		y	y	
54	Ramachandran	45	M		y		RTA 2 wheeler		y		y

55	Balaselvakumaran	19	M		y		RTA 2 wheeler		y		y
56	Sudali	40	M		y		assault		y		
57	Shunmugavel	31	M	y			accidental fall	y			y
58	Muthusamy	49	M		y		assault - bucket		y		y
59	Thangapalam	65	M		y		assault- stone	y			y
60	Kandasamy	76	M		y		RTA 2wheeler		y		
61	Mariselvam	18	M		y		RTA 2wheeler		y		
62	Karim	21	M		y		assault- cricket ball	y			
63	Senthilkumar	27	M		y		RTA 2wheeler	y			y
64	Sankar	22	M			y	RTA 2wheeler		y		
65	Muthukrishnan	23	M			y	RTA 4 wheeler	y		y	
66	John Vincent	36	M	y			RTA 2wheeler	y		y	
67	Sannasi	60	M			y	fall from height		y		
68	Manikandan	17	M	y			RTA 2wheeler		y		
69	Rossammal	43	F	y			acci injury by drumstick	y			y
70	Muthumani	31	M		y		RTA 2wheeler	y		y	
71	Mookammal	50	F		y		assault	y			y
72	Narayanan	13	M	y			assault acci	y			y

73	Puthiavan	45	M		y		RTA 2wheeler		y		
74	Murugan	45	M	y			RTA2wheeler				
75	Ananthakumar	31	M			y	RTA4wheeler		y		
76	Muthupandi	40	M		y		assault hand	y			y
77	palani	29	M			y	assault stone	y			y
78	Sukthar	20	M			y	RTA compression injury chest	y			y
79	Mariappan	20	M			y	RTA2wheeler	y		y	
80	Senthilmeena	22	M		y		RTA2wheeler		y		y
81	Ganesan	39	M	y			acci fall	y			y
82	Kannan	36	M	y			acci injury by stick		y		
83	Rajeshwari	45	F	y			acci injury iron bar	y			
84	Muthusivamani	8	M	y			penetrating injury pencil RE		y		y
85	Kaleeswari	18	F		y		penetrating injury needle RE	y		y	
86	Sakthinarayanan	10	M	y			acci injury stick		y		
87	Pothi	35	M	y			assault stick		y		
88	Subash	12	M		y		acci injury stone	y		y	
89	Srinivasagam	45	M	y			acci injury plastic piece	y			y
90	Sheik seyed	25	M	y			acci injury with stick	y			
91	Xavier	29	M		y		acci injury stick	y			

92	chandramohan	22	M	y			acci injury stick	y			
93	Singarajan	55	M		y		acci injury stone		y		
94	Balasubramanian	25	M	y			acci injury with iron FB		y		y
95	Vijayakumar	35	M	y			acci injury with metal blade		y		
96	Deivanayagam	12	M		y		acci injury needle stick		y		
97	Jebna	11	F	y			injury with tyre		y		y
98	Vijayakumar	11	F	y			injury with stick		y		y
99	Dinesh	16	M	y			RTA 2wheeler	y			y
100	Subbiah	56	M		y		RTA 4 wheeler		y		y